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THE AEROEMBOLISM PROBLEM FOR LONG-RANGE MISSIONS

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FRANKLIN M. HENRY  
AERO MEDICAL LABORATORY

FEBRUARY 1952

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WADC TECHNICAL REPORT 52-84

## THE AEROEMBOLISM PROBLEM FOR LONG-RANGE MISSIONS

*Franklin M. Henry  
Aero Medical Laboratory*

*February 1952*

*RDO No. 696-61*

Wright Air Development Center  
Air Research and Development Command  
United States Air Force  
Wright-Patterson Air Force Base, Ohio

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## FOREWORD

This report was written after and as a direct result of conferences at the Aero Medical Laboratory, Research Division, between the author and personnel of the Wright Air Development Center in which the practical aspects of aeroembolism as a limiting factor in long-range military flights were discussed at length. The purpose of these conferences was to bring together various Wright Air Development specialists in the fields of aircraft engineering, aviation medicine and physiology who then were or later would be especially concerned with this particular aspect of the decompression sickness problem in flight so that they could clearly define the general features and limits of the practical problems involved. It then became the task of the author to evaluate and review the existent physiological data on aeroembolism available in the scientific literature and in various unpublished sources in terms of their applicability to the solution of these practical problems posed by long-range military flights. This review is primarily intended to serve as background information to engineers who are concerned with the design and operation of long-range military aircraft. The author, during the period of formulation and writing of the report, was serving as Special Consultant to the Aero Medical Laboratory under the project identified as RDO 696-61, with J. W. Wilson and Major D. I. Mahoney acting as project engineers.

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**ABSTRACT**

It can be anticipated that cabin pressure differential will be lost in a significant proportion of high-altitude, long-range bombing operations. In such circumstances, it may be necessary to maintain as high an altitude as is consistent with the limitations imposed by the tendency of personnel to develop incapacitating bends, chokes, or other grave symptoms of decompression sickness (aeroembolism).

By breathing undiluted oxygen for several hours under special discipline during the early phase of the flight, at cabin altitudes of the order of 10,000 to 15,000 feet, it is possible to almost completely avoid serious aeroembolism symptoms in later phases that may require cabin altitudes of the order of 35,000 to 38,000 feet, and would otherwise result in 15 to 30% incapacitation. It is shown that the problem becomes particularly acute if all members of the air crew are essential to the mission or to a successful return. Alternative methods of dealing with the situation are considered briefly.

Security Classification of the title of this report is UNCLASSIFIED.

**PUBLICATION REVIEW**

Manuscript Copy of this report has been reviewed and found satisfactory for publication.

FOR THE COMMANDING GENERAL:

*Robert H. Blount*

ROBERT H. BLOUNT  
Colonel, USAF (MC)  
Chief, Aero Medical Laboratory  
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## THE AEROEMBOLISM PROBLEM FOR LONG-RANGE MISSIONS

Although cabin altitudes of 10,000 to 20,000 feet may normally be expected on bomber missions of many hours duration, certain emergency situations are anticipated that will cause or require pressure reduction to 35,000 or 38,000 feet for several hours. A significant proportion of the individuals exposed to these higher altitudes will inevitably develop incapacitating bends, chokes or other grave results of aeroembolism unless preventive measures are instituted. While serious consequences of aviators' aeroembolism have been uncommon in Air Force decompression chambers, this is because of the availability of recompression as a curative procedure. In the emergency situations visualized, recompression by lower altitude flight will not be possible.

It is estimated that without any protection achieved by denitrogenation, 15 to 30% of individuals exposed to the higher altitudes for 1-1/2 hours will suffer serious or complete loss of ability to carry out essential duties. Using the larger figure and assuming that four men have essential duties in the aircraft, 76% of the flights would undergo failure; with the lesser figure, 48% would undergo failure. Tables are available to estimate the proportion of failure with other air crew sizes (see Reference No. 1, Table 1).

Fortunately, flight at cabin altitudes of the order of 10,000 feet prior to reaching aeroembolism-producing altitudes can be expected to have some mitigating influence because of the denitrogenation from pressure reduction. The effect is limited and not linearly cumulative; the chief gain is in the first hour and there is no appreciable gain after the third hour. Using automix at 15,000 and perhaps at 18,000 feet would cause more nitrogen loss and therefore give more protection, but here again the effect is chiefly in the first hour and only a limited gain can be secured. It is probable that exposure to cabin altitudes of the range 20,000 to 25,000 feet prior to subsequent exposure to typical aeroembolism altitudes would be disadvantageous. Above 25,000 feet aeroembolism will occur in proportion to the pressure reduction.

Aeroembolism can be decreased to negligible amounts by increasing denitrogenation through breathing undiluted oxygen for several hours at the lower cabin altitudes prior to high altitude exposure, provided that muscular activity be limited to mild and infrequent exertion. Approximately two hours on oxygen will reduce the amount of aeroembolism to about one-half, another two hours will reduce it to one-fourth and a third consecutive two-hour period will bring it down to about one-eighth the original amount. With more muscular activity, the reduction will be lessened and limited, but it will be considerable and probably be adequate.

Interference with the denitrogenation process by removing the mask to eat or for other reasons, is definitely undesirable, but not disastrous. The loss of protection will increase to the extent that the interruption occurs

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late in the oxygen-breathing period. For example, an aeroembolism rate of 14% after one hour on undiluted oxygen would jump to 16-1/2% after a five-minute interruption and the loss of protection would be regained by 25 additional minutes on oxygen. After four hours on oxygen the rate would have dropped to about 5% and a five-minute interruption would jump it to 9%; the loss of protection would be regained by about two additional hours on oxygen. Obviously the breath should be held if possible when the mask is off. The oxygen discipline should be as strict as if the cabin air were at an unsafe altitude. Mask leakage must be carefully avoided. There will no doubt be some difficulty in enforcement because the cabin will not be at an hypoxic altitude.

When the cabin pressure drops to typical aeroembolism-producing altitudes, such symptoms as do appear are most likely to start within 15 to 45 minutes after the pressure drop, and thereafter the prevailing rate of occurrence will decline by a half during each hour of exposure. Most symptoms will have made their appearance within 1-1/2 hours. Prolongation of the exposure beyond two or three hours will rarely cause new cases to occur, provided that breathing of undiluted oxygen is continued until the aircraft permanently returns to low altitudes.

If the special oxygen discipline is followed, the bends, chokes and other symptoms that do occur can probably be "sat out". After increasing to some peak intensity, symptoms will begin to recede in severity and usually will entirely disappear, although this may require an hour or more. Unnecessary movement or activity, and also hyperventilation (which frequently results from pain and may precipitate syncopal reactions) should be avoided. Emergency medication for relief of "bends" pain has been found ineffective and may cause undesirable side-effects.

On the basis of presently available information, it can be recommended that at least four hours of continuous breathing of undiluted oxygen should precede any anticipated possibility of prolonged emergency exposure to altitudes of the order of 35,000 or 38,000 feet. Particular attention should be given to rigorous oxygen discipline during the last hour or two, to avoid renitrogenation. Six hours might be desirable for certain flight patterns, or to provide a safety factor, although discomfort to personnel and other practical aspects would have to be considered. In addition, the aircraft personnel under consideration should be given an adequate explanation of the reason for the special discipline, the general principles that apply, and the anticipated outcome as outlined in the two preceding paragraphs.

Attention of Flight Surgeons is drawn to the possible desirability of considering extreme susceptibility of an individual to aeroembolism (particularly the "chokes" manifestation) as one of the valid justifications for restricting such an individual from high-altitude flight. While such restriction would certainly not solve the aeroembolism problem and any decision would involve weighing disadvantages, it would help and may indeed prove necessary if the recommended special oxygen discipline cannot be established.

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Supporting references: Development of the basic data and theory, also citation of references, will be found in two Special Reports to the Aero Medical Laboratory by Franklin M. Henry:

- (1) "Aeroembolism in Relation to Exposure Time and Other Factors", 23 June 1951.
- (2) "Anticipated Occurrence of Incapacitating Aeroembolism under Specified Flight Conditions", 12 January 1952.

A collection of monographs summarizing the OSRD-NRC research of 1942 to 1946 has recently been published under the title "Decompression Sickness" (J. F. Fulton, ed., Philadelphia: Saunders, 1951). This book contains a very complete bibliography.

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## APPENDIX I

### AEROEMBOLISM IN RELATION TO EXPOSURE TIME AND OTHER FACTORS

#### SUMMARY

1. For both individual symptom intensity and group incidence, aeroembolism occurrence is the mathematical difference between a growth factor (dependent on pressure differential and amount of physical activity) and a decay factor (identified with the tissue denitrogenation process). It follows that typical aeroembolism is a self-limited disease--if the peak symptoms can somehow be resisted successfully, recovery and immunity for the course of a particular decompression can be expected provided that renitrogenation does not occur. After the first hour or two of exposure, there is no further accumulation of symptoms.
2. While considerable protection will be secured by breathing low-nitrogen gas mixtures (e.g., automix during flight), a high protection rate requires breathing pure oxygen during the later stages of denitrogenation, and is achieved more slowly than commonly realized.
3. Provision of some sort of temporary recompression device for affected personnel seems indicated, both on psychological grounds and to cope with the anticipated failures of denitrogenation prophylaxis.
4. Tabled probability values are presented to illustrate the advantages of interchangeability of essential duties during flight, and the increasing hazard as the size of an air crew is increased. Preselection is discussed briefly.

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## TEMPORAL COURSE AND SEVERITY OF AEROEMBOLISM

Group incidence. It has been observed that symptoms of aeroembolism do not accumulate at a uniform rate--on the contrary, during the early stages of decompression there are few symptoms. The rate of appearance of symptoms (or for that matter, of incapacitation) increases, reaches a maximum, and then declines. The course of events may be described mathematically by postulating exponential growth and decay factors (1), leading to the expression

$$dy/dt = a_1 e^{-k_1 t} - a_2 e^{-k_2 t}$$

where the first subscript refers to the decay factor and the second refers to the growth factor. It is found that decompression chamber data are satisfactorily described by this formula, at least for the first 1-1/2 hours of exposure, and when the number of individuals tested is sufficiently large to justify an attempt at curve fitting.

Testing of the adequacy of the formula has been largely limited to onset of symptoms, rather than incapacitation, under the reasonable assumption that both represent stages of the same fundamental process, with the former yielding much more extensive data. A more specific theoretical identification of the curve constants, derived from an equation describing the growth and decay of a single bubble, has subsequently become available (2, page 13).

In Figure 1 is shown a family of curves based on the above formula, with the curve constants derived from University of California data, involving two chamber runs on from 60 to 80 individuals for each curve. The experimental variable in this case is severity of exercise; with increasing amounts of activity the velocity constant and the intercept (i.e., amount) of the growth factor appears to increase, whereas the velocity constant of the decay factor is relatively constant and has the magnitude of known velocity constants of denitrogenation as established by direct measurement. Curve A, involving almost no exercise, appears to constitute an exception, but this is explainable on the basis of known facts concerning denitrogenation, as follows:

It is an oversimplification to represent denitrogenation (i.e., the decay factor) as a single exponential term--actually, the decay factor should be described as the sum of a series of exponentials. The first, a very rapid component, is of no consequence in aeroembolism; the second or visceral and muscle-connective tissue component is important up to about 90 minutes, and thereafter the fat component, which is quite slow, is the major factor (2, pages 83, 92 and elsewhere). The data for curve A are not sufficiently extensive as to length of time or number of individuals to measure the slow "fat" component directly with any precision, however, it must have been present and would account for the apparently slower decay.

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Figure 2 shows another family of curves. In this case the experimental variable is altitude, ranging from 30,000 to 38,000 feet. The denitrogenation factor is noticeably faster at the higher altitudes, in agreement with direct observation (3, page 55), although the main difference is in the direction of a larger and faster growth factor.

The implication of the data of Figures 1 and 2 would seem to be:

- a. In situations involving no preflight denitrogenation the peak rate of symptom incidence is reached after 10 or 15 minutes of exposure if the altitude is high and the amount of muscular activity is large. Thereafter, the rate declines to the extent of 50% per 15 minutes of exposure (approximately).
- b. At lower altitudes or with a minimum of physical activity, the peak rate is not reached until after some 30 minutes of exposure, and the rate declines more slowly, requiring 25 or 30 minutes to drop 50%.
- c. The influence of degree of physical activity, while definite, has less effect than is commonly thought to be the case. There are other lines of evidence that agree with this observation (4).
- d. After the first hour or two of decompression, the appearance of new cases of symptoms should be uncommon, although rare cases will surely occur even after several hours of exposure since the denitrogenation curve is exponential and has several components of successively slower rates.

Individual symptoms. It is a common observation that the pain of "bends" may increase to some maximum amount and thereafter partially or completely retrogress, or on the other hand may continue to increase to an intolerable degree and result in incapacitation. Extensive observation has established that the typical unbearable pain rises in intensity 3-1/2 times more rapidly than the symptom that is fated for partial or complete regression. Furthermore, the empirical time-intensity curve for the latter type of symptoms is observed to be concave downward in trend even before regression becomes clearly apparent, whereas the former (intolerable) type is characterized by a linear or slightly upward curvature (5). While no attempt to fit a mathematical equation to pain curves has been reported, there is little question but that the salient features are described by the Nims theoretical formula postulating exponential growth and decay of an "ideal" bubble (2, page 12 and his Figure 8). Individual curves as observed in the decompression chamber are of course somewhat irregular and sometimes atypical, but it certainly does not require a very strong imagination to visualize them as representing the above-threshold portion of the theoretical growth-decay function (see Figures 3 and 4).

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Inspection of individual decompression chamber symptoms leads to the impression that even the severe symptoms would ultimately exhibit downward concavity, and regress, if the pain could be withstood or if it did not result in secondary or other effects of serious consequence. For obvious reasons, data bearing directly on this question are neither systematic nor extensive, and while instances of successfully "sitting out" the bends are known (6, page 303) it is impossible to make a recommendation at the present time. Nevertheless, it is suggestive that if one accepts the theory that aeroembolism is due to gas bubble expansion, "bends" should regress as a result of continued denitrogenation and equilibration if the peak symptom intensity can once be passed. Theoretically, this critical pre-regression phase should occur and should be passed, even if the ambient altitude is lowered sufficiently to relieve the pain. Unfortunately, there seems to be no very direct experimental evidence from human decompression chamber experiments to test this hypothesis, although the group data from the Canadian experiments on "step-wise ascents" (7) is certainly in its favor.

Theoretically, "chokes" should also regress after passing a critical peak phase. Crucial data are lacking because this aspect of aeroembolism is considered too dangerous to justify prolonged experimental observation (8).

Maximum tolerable symptoms: Psychological factors: No definitive answer can at present be made to the question of just what proportion of aeroembolism symptoms will result in aborted or ineffective missions under combat conditions. It is entirely possible that such an answer will never be available. In decompression chamber experiments, the practice has been to observe symptoms to some arbitrary standard of incapacitation that is considerably (and understandably) less severe than complete collapse at altitude. Nevertheless, cases of collapse have been reported in decompression chambers (9, 10) as well as in aircraft (6). It is probably significant that the highest incidence of syncopal reactions--in 16% of chamber runs--occurred in a project that was conceded by other research groups to carry the subjects further toward an ultimate end point than was typical of most decompression chamber experiments (9).

On the other hand, the writer has on a number of occasions observed or personally experienced situations in which a bends symptom that was to all intents and purposes quite incapacitating, did not prevent the carrying out of such motor activity as was essential to cope with (for a short period of time) an emergency situation that developed in the decompression chamber. While such reports do not appear in the literature, it is believed that others have had comparable experiences. Furthermore, it is thought by a number of experienced decompression chamber personnel that submission to repeated exposure to the certainty of severe bends is possible only because the subject can, under experimental conditions, secure escape from the situation in short order at his own volition, whenever he so desires.

While the significance of these psychological factors cannot be evaluated quantitatively, it seems highly probable that if some sort of "bends bag" is available to members of the air crew, it would ordinarily be possible to carry out essential duties (at least on a short-time basis) while suffering from pain so severe that the situation would prove psychologically intolerable

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if no possibility of relief were in sight. Certain other severe symptoms of aeroembolism would presumably not be made any less incapacitating except while actually under recompression in the bag, but these are considerably less frequent than typical bends (6, pp., 298-9, and several other published reports).

It would seem desirable to consider the psychological factors in the precombat indoctrination of air crew (this term being intended to include the entire flight personnel of an aircraft). Without minimizing the potential gravity of aeroembolism, the facts as to the probability of regression of symptoms after passing the crucial maximum period can be emphasized; the nonregressive symptoms may be taken as a calculated risk comparable to the hazard of enemy attack. The California decompression chamber experience is that from 29 to 50% of individuals having symptoms of aeroembolism at 38,000 feet will be protected by symptom regression; at 30,000 feet the figure is 72 to 74%. There is reason to believe that these figures are very conservative because the criterion for recompression in these operations was 100% safety for the individual under exposure; therefore, in the face of ignorance concerning just how far the symptom severity could be allowed to increase without undue hazard, the experiments were terminated at a relatively early stage in the development of the bends pain.

### EFFECTIVENESS OF PREOXYGENATION

Variability. Widely variant results have been reported from decompression chamber experiments on the effectiveness of pre-exposure denitrogenation. Nims, for example, analyzes data from four laboratories to show that from 15 minutes to two hours are required for 50% protection (2, Figure 11). Jones has examined additional data from other laboratories, finding similar variations which he ascribes to the age factor. It may be seen by inspection, however, that this concept furnishes only a partial answer (3, page 91). In most cases the groups studied have been small, so that sampling error is large, and relatively short periods of denitrogenation have been used.

A further factor causing confusion has been that some of the earlier experiments on preoxygenation were done before the temporal course of symptom appearance had been worked out. These particular experiments involved only a short exposure to altitude, leading to the erroneous conclusion that short periods of denitrogenation would yield nearly perfect protection.

Significance of slow component. Inert gas exchange curves plotted as a function of time (3, page 96), particularly when considered with reference to such data as Figures 1 and 2 of the present report, show why protection cannot be secured so quickly. Some of the California chamber series involved one and two hours of preoxygenation of sizable groups of individuals (60 or more), permitting the plotting of the time course of symptom development as a function of preoxygenation time and altitude exposure. These data

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are shown in Figure 4, together with a superposed typical denitrogenation curve obtained by direct measurement (3, page 82). There is an obviously similar trend in the two types of data, implying that denitrogenation will progress much more slowly after the first 1-1/2 or two hours of oxygen breathing.

Time required for protection. Direct data on the effectiveness of very long periods of denitrogenation--five to eight hours--are too few to warrant any direct conclusions that such procedure will or will not result in complete protection. It is known that four hours will not give complete protection, but since the severity as well as the incidence of aeroembolism is reduced by the procedure, true incapacitation is rare with this amount of prophylaxis (6, 12).

Tables giving the amount of protection to be expected with varying amounts of denitrogenation are available for both pure oxygen and automix supply (3, page 97 ff.). These tables should prove dependable up to three or perhaps four hours, since they are based on decompression chamber experience within these limits. They do not take into account the importance of slow-component denitrogenation for longer times, hence, it would be safer to at least double the required time increments beyond the above range. It should be noted that when using automix at 20,000 feet, it is theoretically impossible to secure more than 70 to 75% protection because of residual alveolar  $pN_2$ . At 25,000 feet, four to five hours should result in 85 to 90% protection for subsequent exposure to higher altitude, with the risk of one or two chances per 100 of incapacitation at 25,000 feet. To secure greater protection from in-flight denitrogenation, 100% oxygen (nominal) must be substituted for automix at 20,000 feet and care must be taken to prevent mask leakage.

It should be noted that a low alveolar  $pN_2$  is most important in the late, rather than early, stages of denitrogenation. For example, using available tables (3, page 98), one-half hour of air-breathing results in a loss of three protection units when it follows one hour of preoxygenation, whereas after two hours of preoxygenation the loss is 10 points, and after four hours it is 23 points, dropping the protection from 75% to only 52%. The loss is only 90% as large if the air is breathed at 10,000 feet rather than sea level, but the same principle applies (3, page 100).

### ALLOWABLE INCAPACITATION RATE: REQUIRED PROPHYLAXIS

In the last analysis, the allowable abortion rate is a matter of judgment. Reasoning from the "Law of least squares" familiar in error analysis, it would seem that any specific potential cause of failure should be kept as low as one-fourth the next greatest cause; further reduction will have only a minor effect on total loss (since errors add up as the square root of the sum of squares), although if an additional reduction can be made easily and cheaply, it should certainly be accomplished.

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Probably a 1% loss factor is not serious; a 5% loss may or may not be depending on the other factors. Inspection of Table I will show that the required freedom from flight abortion depends on the specific air-crew design as well as individual freedom from disability. To hold the abortion rate to 1% would require nearly complete individual prophylaxis. If a 5% rate is allowable, the demands are not quite so rigorous, but they are certainly high. Even a 10% figure is not easily attained. However, it should be kept in mind that if only 10% of the aircraft on a mission are exposed to aeroembolism conditions, and 10% of these abort, the total abortion rate is only 1%.

If a typical flight be visualized as subjecting the air crew to about 25,000 feet for several hours followed by exposure to 35,000 or 40,000 feet, a protection of the order of 85 or 90% for the higher altitude may be anticipated. This might be considered adequate if individual incapacitation without the protection is no more than say 10%. From what has been said earlier, it is clear that flights at 30,000 feet could be accomplished, but the abortion or nonreturn rate would probably be too large at higher altitudes (see Figure 6). Introduction of 100% oxygen breathing would appear to be necessary. Since it may not solve the problem completely, it would seem that some development of the "bends bag" might well be of crucial importance; the desirability of breathing oxygen while recompressed should be considered in its design.

Preselection. It will be noted that the calculations for Table I assumed equal susceptibility for all crew members. While this assumption does not seriously affect the results, it does point up the fact that the presence of a particularly susceptible individual in a certain air crew greatly increases the hazard for that particular aircraft. It should also be mentioned that older individuals are in general much more susceptible, especially to the more severe symptoms such as chokes, and achieve denitrogenation more slowly than younger men (3). It is likely that an individual of greatest experience is apt to be older. The incapacitation of such an individual might well prove particularly costly.

There is no question at all that individual susceptibility exists and that highly susceptible individuals are far more likely to suffer from incapacitating aeroembolism than is the case with others (6), although the relationship is not perfect. While a truly resistant group cannot be insured by preselection, it is also true that eliminating say the 10% who are most susceptible will result in a worthwhile gain, and can be justified if not too costly in other respects. There is little further gain from eliminating more than the extremely susceptible individuals. With respect to interpretation, it should be emphasized that the increase of probability of success from, for example, 90% to 95% may not sound sensational, but it does in fact indicate that the severity of the problem has been cut in half, which is certainly a considerable gain.

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## APPENDIX I

TABLE I

PROBABILITY OF COMPLETING A MISSION  
as a function of

- (a) Individual probability of avoiding casualty.
- (b) Number of essential crew positions.
- (c) Size of air crew.
- (d) Possible replacement of casualty by nonessential number.

Individual probability	.95	.90	.85	.80	.75	.70	.65	.60	.55	.50
<b>TWO ESSENTIAL</b>										
Crew of 2 -----	.90	.81	.72	.64	.56	.49	.42	.36	.30	.25
Crew of 3 (replace one)	.95	.89	.83	.77	.70	.64	.57	.50	.44	.38
Crew of 3 (replace any)	.99+	.97	.94	.90	.84	.78	.72	.65	.57	.50
Crew of 4 (standby for each of two)	1.00-	.98	.96	.92	.88	.83	.77	.71	.64	.51
<b>THREE ESSENTIAL</b>										
Crew of 3 -----	.86	.73	.61	.51	.42	.34	.28	.22	.17	.13
Crew of 4 (replace one)	.90	.80	.71	.62	.53	.45	.37	.30	.24	.19
Crew of 4 (replace two)	.95	.88	.80	.72	.63	.55	.47	.39	.32	.25
Crew of 4 (replace any)	.99	.95	.89	.82	.74	.65	.56	.48	.39	.31
Crew of 5 (standby for each of two)	.95	.88	.81	.74	.66	.58	.50	.41	.35	.28
Crew of 6 (standby for each of three)	.99	.97	.93	.89	.82	.75	.67	.59	.51	.42
<b>FOUR ESSENTIAL</b>										
Crew of 4 -----	.82	.66	.52	.41	.32	.24	.18	.13	.09	.06
Crew of 5 (replace one)	.86	.72	.60	.49	.40	.31	.24	.18	.13	.09
Crew of 5 (replace two)	.90	.79	.68	.57	.48	.38	.30	.23	.17	.13
Crew of 5 (replace three)	.94	.85	.76	.66	.55	.46	.37	.29	.22	.16
Crew of 5 (replace any)	.97	.92	.83	.74	.63	.53	.43	.34	.26	.19
Crew of 6 (standby for each of two)	.90	.79	.68	.57	.48	.38	.30	.23	.17	.13

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TABLE I (cont'd)

Comment on Probability Table:

1. The Table is based on expansion of the binomial  $(p+q)^n$ , subtracting terms or parts of terms that fail to meet the requirements of the mission. While not mathematically rigorous because equal probabilities are assumed for all members of a crew, it is believed to represent a reasonable engineering-type of estimate that will prove dependable when applied to the average of a large number of flights.
2. Several facts revealed by the Table should be noted.
  - (a) The hazard increases rapidly as individual probability of success drops off.
  - (b) This effect is considerably enhanced as the number of essential positions increases.
  - (c) Probability of success increases markedly if replacements are available.
  - (d) This gain is more difficult to achieve as the number of essential positions increases.
  - (e) Versatility of replacements is of great value.
3. General evaluation by the writer is, that with a small essential air crew and some versatility, the problem is not great if individual probability can be kept fairly high, approaching 0.95. With larger air crews, the problem is potentially quite serious, and will no doubt require a combination of several methods to insure a very high individual probability of success.

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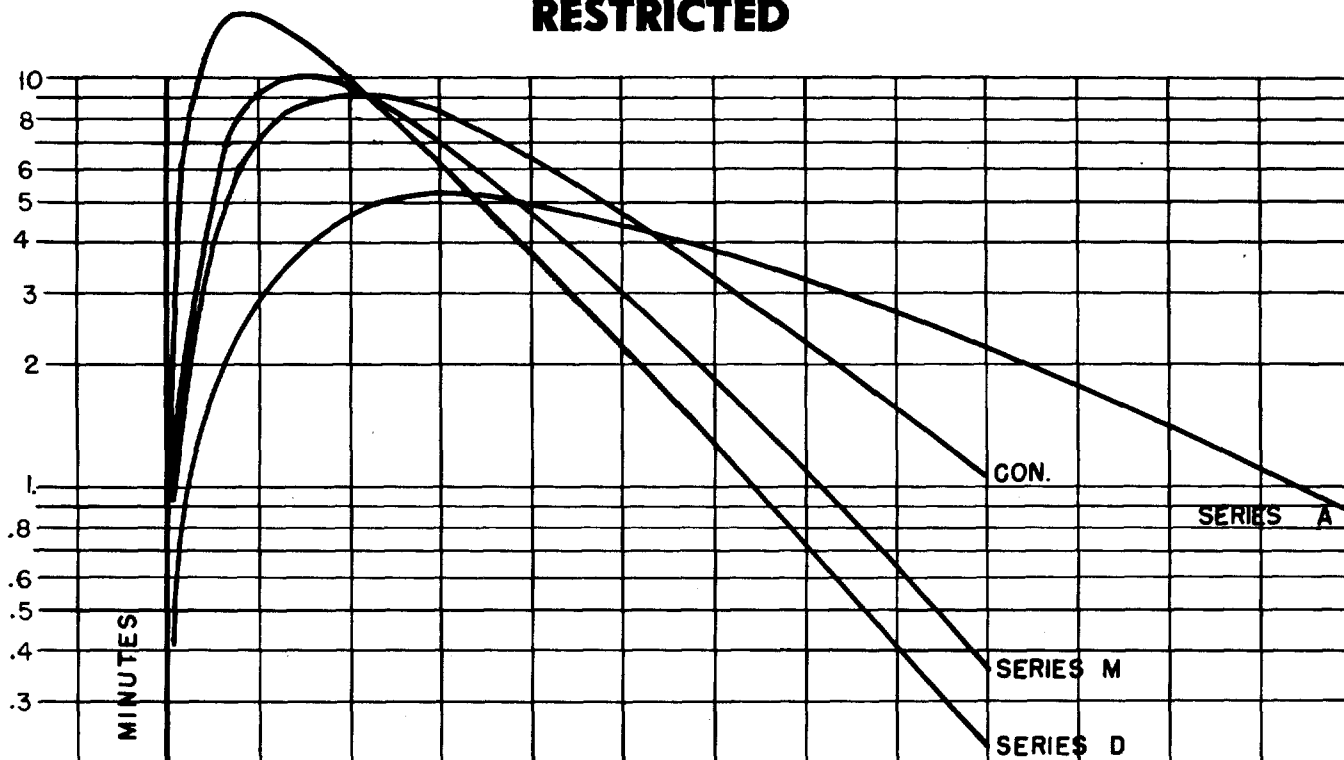


FIG. 1. EFFECT OF WIDE VARIATION OF EXERCISE ON SYMPTOMS. (SERIES A, -- STAND UP TWICE, ONE KNEE BEND, THREE ARM THRUSTS EACH 10 MIN; M, 10 STEP-UPS EACH 5 MIN; D, SAME, EACH  $2\frac{1}{2}$  MIN.)

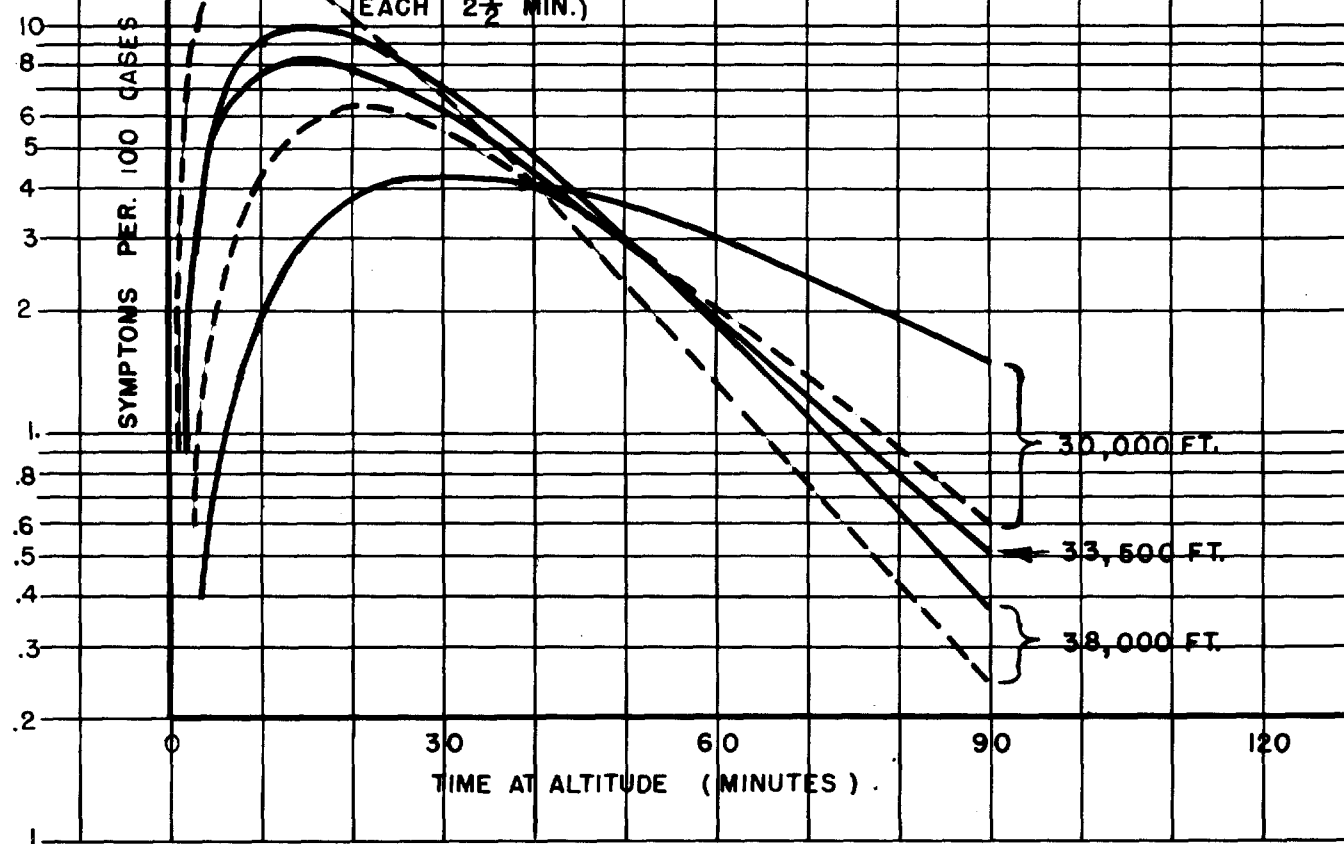


FIG. 2. EFFECT OF ALTITUDE ON SYMPTOM INCIDENCE (SOLID LINES, EXERCISE AT 5 MIN. INTERVALS; DOTTED, EXERCISE AT  $2\frac{1}{2}$  MIN. INTERVALS)

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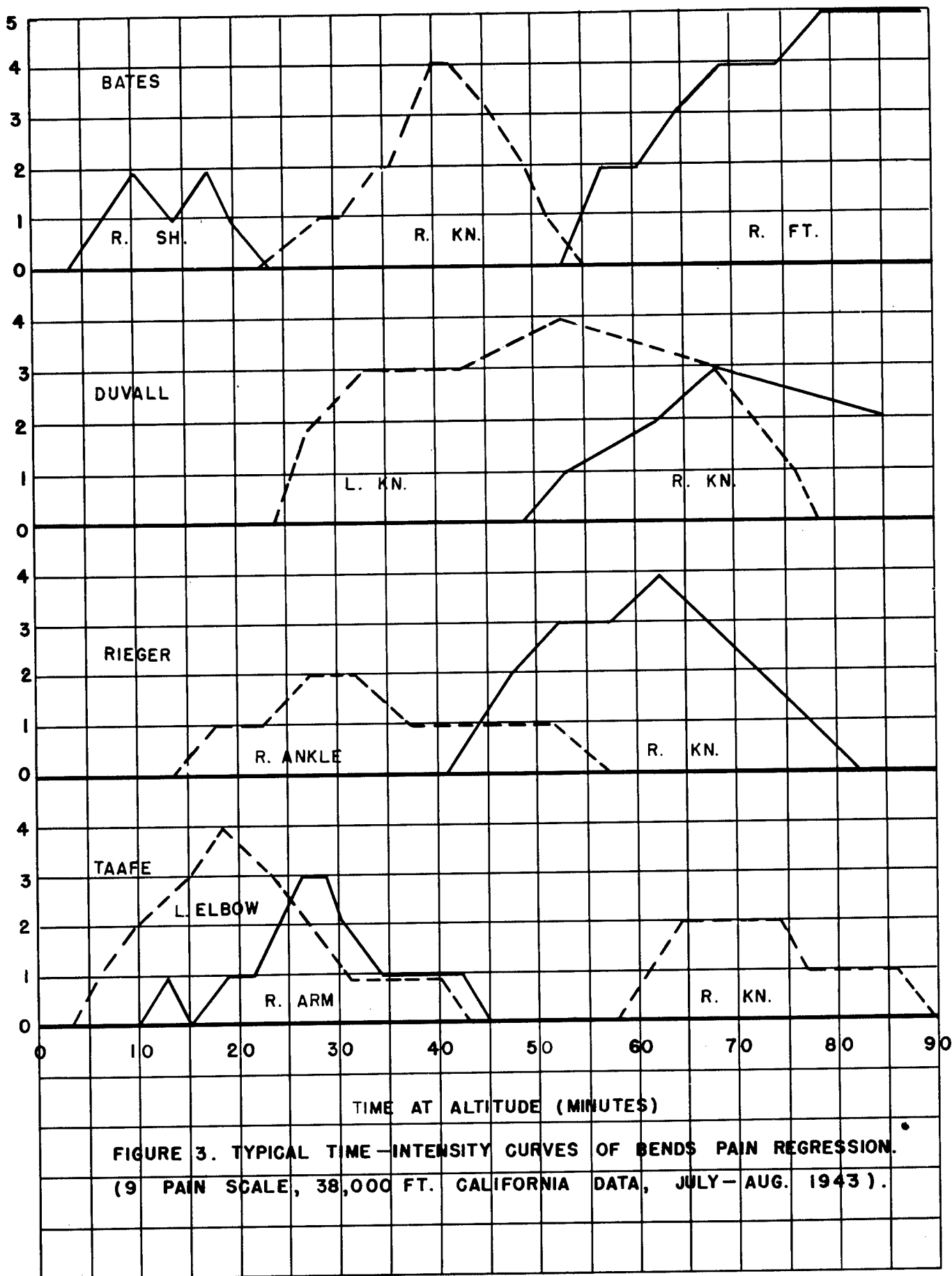


FIGURE 3. TYPICAL TIME-INTENSITY CURVES OF BENDS PAIN REGRESSION.  
(9 PAIN SCALE, 38,000 FT. CALIFORNIA DATA, JULY-AUG. 1943).

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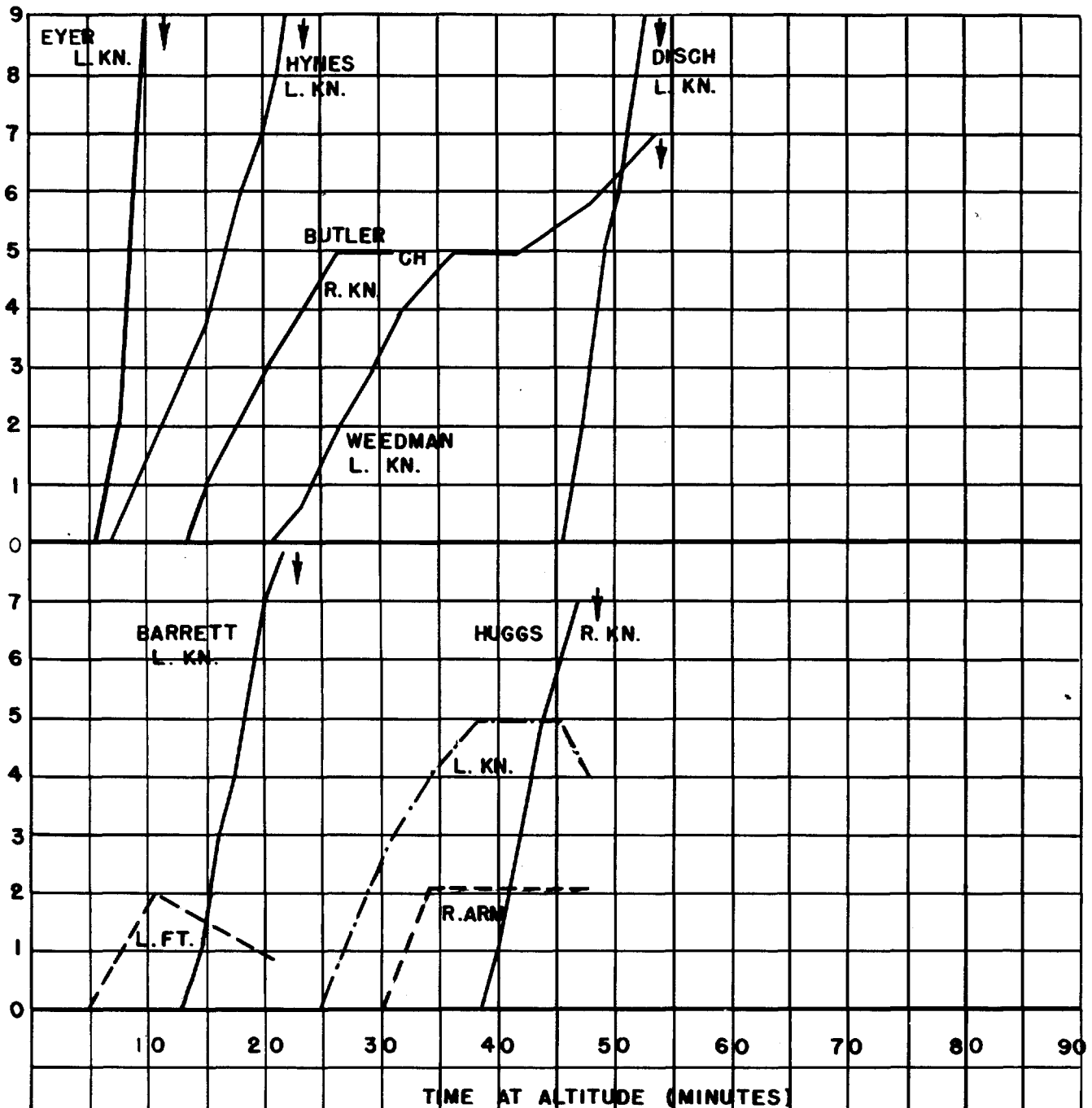
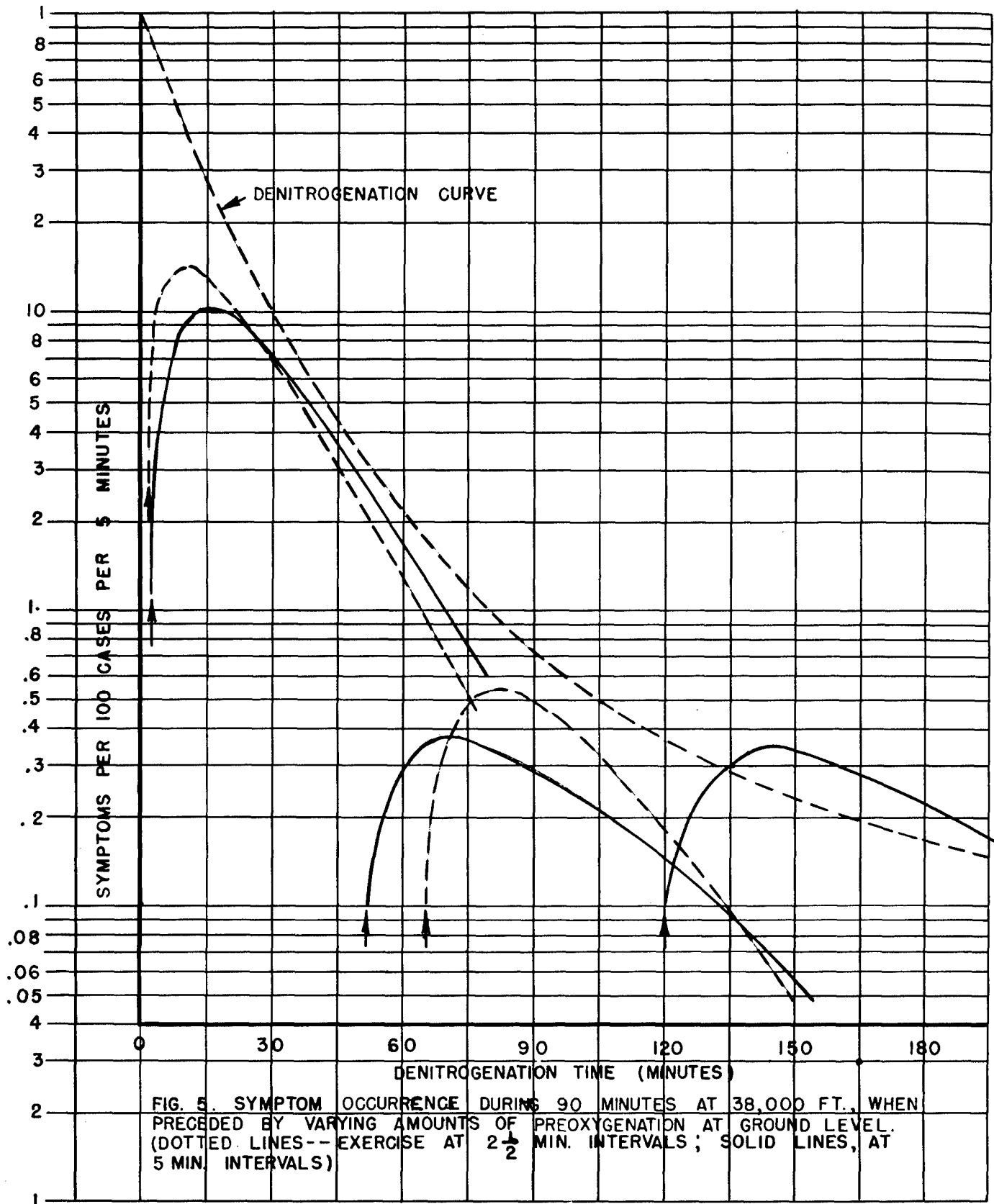


FIGURE 4. TYPICAL TIME-INTENSITY CURVES OF INCAPACITATING BENDS PAIN. (38,000 FT. CALIFORNIA DATA, JULY-AUG. 1943)

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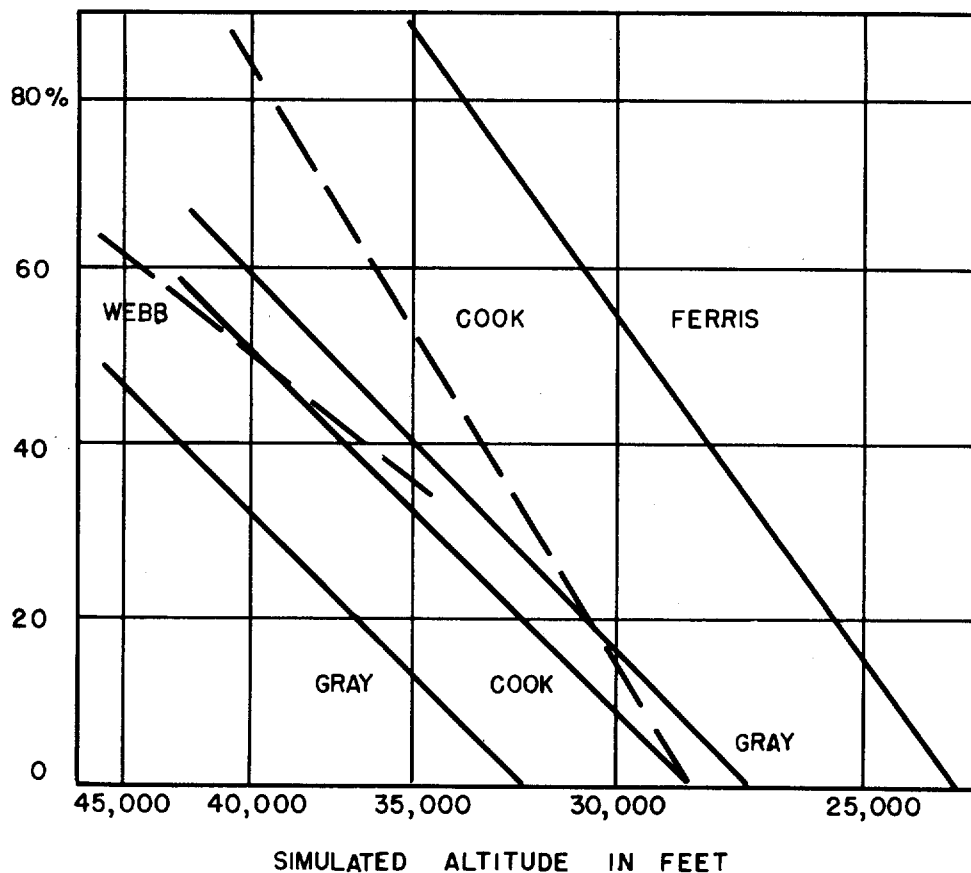


FIG. 6. AEROEMBOLISM INCIDENCE AS A FUNCTION OF ALTITUDE  
(AFTER NIMS, 2 ).

NOTE: IT IS IMPOSSIBLE TO CONSTRUCT A GRAPH LIKE THIS FOR INCAPACITATION, DUE TO VARYING STANDARDS. IT HAS BEEN REPORTED THAT 10 PERCENT OF SYMPTOMS AT 25,000-30,000 FT. ARE INTOLERABLE IN OPERATIONAL FLYING (SMEDAL & HALL, U. S. N. MED. NEWS LETTER, NOV. 1948). COMPARED WITH 25 PERCENT IN THE CALIFORNIA DECOMPRESSION CHAMBER. AT 38,000 FT. IN CHAMBERS, TYPICAL FIGURES ARE AS HIGH AS 50-60 PERCENT.

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## APPENDIX II

ANTICIPATED OCCURRENCE OF INCAPACITATING AEROEMBOLISM  
UNDER SPECIFIED FLIGHT CONDITIONS

1. The undenitrogenated rate of individual incapacitation during an exposure of three hours at 35,000 to 38,000 feet with moderate physical activity is estimated to be 29%.
2. It is assumed that personnel will be exposed to cabin altitudes of the order of 10,000 feet for specified periods of time, followed by
  - (a) Exposure to 35,000 - 38,000 feet for several hours, or
  - (b) Continuation for several hours at 10,000 to 20,000 feet breathing undiluted oxygen, followed by exposure as in (a) above.
3. The calculated percentage of exposed individuals who will become incapacitated from aeroembolism under various permutations of these variables, is set forth below.

(In this Table, the symbol 10 M implies exposure without supplemental oxygen at 10,000 feet during the designated block of flight time. The symbol O<sub>2</sub> implies exposure at 15,000 feet or less on undiluted oxygen for the block time. The percentage figures refer to the anticipated aeroembolism during 1-1/2 blocks of time at 35,000 to 38,000 feet. Reducing this exposure to one block or lengthening it to many blocks will not materially affect the aeroembolism amount).

0 - 4 hrs	4 - 6 hrs	6 - 8 hrs	8 - 10 hrs	10 - 12 hrs	12 - 14 hrs
10 M	19% to 21%				
10 M	10 M	19% to 20%			
10 M	10 M	10 M	18% to 19%		
10 M	10 M	10 M	10 M	18% to 19%	
10 M	10 M	10 M	10 M	O <sub>2</sub>	5% to 9%
10 M	10 M	10 M	O <sub>2</sub>	5% to 9%	
10 M	10 M	O <sub>2</sub>	6% to 10%		
10 M	10 M	O <sub>2</sub>	O <sub>2</sub>	2% to 5%	
10 M	O <sub>2</sub>	O <sub>2</sub>	O <sub>2</sub>	0.1% to 0.3%	

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4. Certain qualifications should be mentioned. (Most of these were discussed in a Special Report dated 23 June 1951, to the Aero Medical Laboratory from Franklin M. Henry, "Aeroembolism in relation to exposure time and other factors").

The incidence of incapacitation in actual emergency conditions during operations may be only half (approximately) of the estimation. There are other factors, difficult to evaluate, that would tend to raise it. Although the allowable individual casualty rate from aeroembolism depends on the indispensability of particular air-crew positions, it would seem necessary to hold it to less than 5% and desirable to keep it down to 1%.

The calculations represent statistical averages, and operationally observed rates are sure to vary considerably in both directions in samples of the order of 10 to 100 flights.

5. (a) A conservative estimate places the minimum oxygen allowance as sufficient for four hours of undiluted supply during cabin altitudes of less than 20,000 feet prior to the possible increasing of cabin altitudes to 35,000 or 38,000 feet for any appreciable time, in addition to such other allowance as may be necessary for anticipated operations. While this allowance does not provide for much of a "safety factor", it will probably be enough to get by with.

(b) Should the flight plan contemplate from two to four hours at cabin altitudes of 25,000 to 28,000 feet between the 10,000 to 20,000 foot time blocks and possible cabin altitudes of 35,000 - 38,000 feet, provision should be made for an additional two hours on undiluted oxygen at the lower altitude.

6. Provision of the recommended oxygen supply must be implemented by enforcement of the recommended oxygen discipline or the predicted freedom from aeroembolism casualties will not materialize. Some difficulty in securing cooperation may be anticipated since most flights (it may be hoped) will not require the 35,000 - 38,000 foot phase, and the outcome of those that do will be conditioned by many factors.

7. For sources of basic data and method, see Appendix III.

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### APPENDIX III

Incidence of Incapacitating Aeroembolism in Unprotected Personnel: The following gross figures are available for decompression chamber experience:

NAF - 14%

RCAF 24%

California 38%

AAF - 15%

Yale 35%

Cincinnati 45%

The unweighted average is, in round numbers, 29%. Data from any one source are extremely variable; for example, AAF figures from five locations range from 5 to 38%. The number of individuals tested was large--about 45,000 for the AAF, several thousand for the Canadian data, about 1000 for NAF and the same for combined civilian chambers. In general the military exposures were without exercise while the civilian experiments involved regular exercise. Altitudes of 35,000 and 38,000 feet were involved.

These data are found in various NRC (U.S.) and NRC (Canada) reports, and some reports in summarized form in the Monograph Decompression Sickness, J. F. Fulton, ed., Saunders, 1951.

Denitrogenation Rates and Methodology for Calculating Protection. See Studies on Gas Exchange, MCREXD - 696 - 114, Aero Medical Laboratory. Memorandum Report, 1948 (Reprinted in Decompression Sickness Chapter IX, Part II. Essentially the same theory is also found in Chapter VIII, p. 220.)

Another method for calculating protection has been proposed (see Decompression Sickness, Chapter IX, Part I). This method was not used because validation of the theory involved extensive discarding of data that did not fit.

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